# Ruptured abdominal aortic aneurysms: Factors affecting mortality rates

Linda M. Harris, MD, Gian Luca Faggioli, MD, Roger Fiedler, PhD, G. Richard Curl, MD, and John J. Ricotta, MD, Buffalo, N.Y.

Outcome of 113 operations for ruptured abdominal aortic aneurysms were reviewed to determine the contribution of perioperative events to mortality rates. Preoperative, intraoperative, and postoperative factors were examined with regard to their influence on early and late deaths. A mortality rate of 64% (72/113) was unrelated to age, gender, and preexistent medical conditions. Death within 48 hours occurred in 42 of 72 patients (58%). Preoperative status, including cardiac arrest, loss of consciousness, and acidosis influenced early deaths (<48 hours) but not late deaths. Early deaths were also influenced by severe operative hypotension and excessive transfusion requirements. Late deaths (>48 hours) occurred in 30/72 cases (42%) at a mean of 24.6 ± 22.9 days. Late death was related to postoperative organ system failure, specifically renal and respiratory failure, and the need for reoperation. The overall mortality rate was influenced by preoperative, intraoperative, and postoperative factors. Postoperative renal failure was the strongest predictor of overall deaths. Survival after ruptured abdominal aortic aneurysm depends on intraoperative and postoperative complications as well as preoperative conditions. Late death, the greatest strain on resources, is independent of preoperative status. The thesis that some patients with ruptured abdominal aortic aneurysm should be denied operation to conserve resources is not supported by these data. Efforts to improve survival should focus on reducing intraoperative complications and improving management of postoperative organ failure. (J VASC SURG 1991;14:812-20.)

Deaths from elective abdominal aortic aneurysm (AAA) repair have been dramatically reduced since the initial repairs by Dubost et al.<sup>1</sup> and Gaylis and Kessler<sup>3</sup> in 1951; however, aneurysmectomy for ruptured aneurysms continues to be associated with high mortality rates in the 50% to 70% range. If prehospital deaths are considered, the mortality rate rises to greater than 80%.<sup>3-5</sup>

Despite the increase in numbers of elective AAA repair, the number of patients with ruptured abdominal aortic aneurysm (RAAA) has not been significantly reduced.<sup>4,6,7</sup> The persistent high mortality rates after rupture and emergency repair have led to the assumption that the patient's preoperative condition may predetermine his survival. In turn this has led some authors to suggest that subgroups of patients might be excluded from operative consideration based on excessive operative mortality rates associated with certain preoperative characteristics. The ethical implications of this are profound. Before accepting such a position we sought to define the relative importance of preoperative patient status, intraoperative events, and postoperative complications on mortality rates. Although most studies evaluate operative and postoperative mortality rates separately, rarely is a distinction made between early and late postoperative death. Since a prolonged postoperative course with eventual death has the greatest negative economic impact, we believed that looking at early and late death separately would be important. The questions asked in this study were four: (1) What are the factors associated with excessive morbidity and mortality rates after surgery for ruptured AAA. (2) What are the relative contributions of preoperative, intraoperative, and postoperative complications to overall morbidity and mortality rates from RAAA? (3) How to determine whether nonoperative management of some patients could be justified based on their preoperative condition. (4) What savings in resources might result from such a policy? We hoped that answering these questions would help determine whether or not the premise that some

From State University of New York at Buffalo Department of Surgery.

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Reprint requests: John J. Ricotta, MD, Department of Surgery, Millard Fillmore Hospital, 3 Gates Circle, Buffalo, NY 14209. 24/6/33494

patients can be denied operation based on a protocol could be supported.

# **METHODS**

A retrospective study was performed including all patients with infrarenal RAAA at two of the teaching hospitals of SUNY at Buffalo between 1980 and 1989. Expanding or symptomatic aneurysms that had not ruptured were excluded. Charts were reviewed with regard to the patient's preoperative status, status on arrival in the emergency department, and intraoperative and postoperative complications. Preexisting medical conditions evaluated included the following: a history of cardiac, pulmonary, renal, or vascular diseases, obesity, previous abdominal surgery, and the known presence of an AAA. Length of time from onset of symptoms to arrival in the hospital, and time to operation were recorded. Data gathered on hospital arrival included vital signs, hemoglobin, arterial blood gases, electrolytes, neurologic condition, and the presence of a preoperative cardiac arrest. After operation, data were gathered on the occurrence of renal, respiratory, cardiac, and neurologic complications, lower extremity ischemia, bowel ischemia, and the need for reoperation. Length of stay in the intensive care unit (ICU), duration of ventilatory support, and intraoperative and postoperative transfusion requirements were recorded.

Multivariate statistical analysis with logistic regression was performed. Chi square and one-way analysis of variance (ANOVA) were also used, as appropriate, to correlate overall, early, and late deaths with examined factors.

Postoperative deaths appeared to occur in a bimodal distribution, either early after operation or 1 or more weeks after operation. Therefore for the purpose of analysis, death was defined as early, within 48 hours of operation, including operative deaths; and defined as late, greater than 48 hours after operation. Patients who died before operation or in whom permission for operation was denied were excluded from the statistical analysis. All patients not operated on died.

Renal failure was defined as serum creatinine > 3.0 mg/dl. It was further subdivided into anuric, < 100 ml/24 hours; oliguric, 100 to 600 ml/24 hours; and high output, > 600 ml/24 hours. Respiratory failure was defined as greater than 7 days of ventilatory support, pneumonia, adult respiratory distress syndrome, or the need for tracheostomy. Cardiac failure included myocardial infarction and cardiogenic shock. Neurologic complications included cerebrovascular accident and spinal cord

<b>Fable</b>	I.	Demographics	
		L'onnographico	

Characteristic	Total	Nonsurvivors	Survivors	
Age	$72.8 \pm 8.1$ (50-89)	$73.2 \pm 8.0$	$72.2 \pm 8.0$	
Gender	Àale 93	Male 61	Male 32	
	Female 20	Female 11	Female 9	
Preexisting medi-				
cal conditions				
Pulmonary	50	29	21	
Cardiac	53	31	22	
Peripheral vas- cular	22	16	6	
Hypertension	57	36	21	
Diabetes mel- litus	7	3	4	
Cerebrovascular	17	12	5	
Obesity	17	10	7	
Previous ab- dominal surgery	34	24	10	

ischemia. Occurrence of lower extremity ischemia and bowel ischemia was identified by the need for operative intervention.

# RESULTS

One hundred twenty-six patients were admitted with infrarenal RAAA. Thirteen patients did not undergo operative intervention because of the wishes of the patient's family, leaving 113 patients for analysis. Patients who were not operated on were generally older than the group operated on; however, no one was denied operation based on age. Demographics of the population are listed in Table I.

Age ranged from 50 to 89 years. Sixteen of the 26 patients over 80 years old died (62%), and 57 of the 87 patients less than 80 years old died (66%).

Twenty-six patients died in the operating room, and 87 survived operation. Forty-six of these 87 patients died after operation, and 20 survived but had major complications. Only 25 patients (29%) had no postoperative problems. No primary aortoenteric fistula was identified in this population.

Six patients were incorrectly diagnosed and sustained delays in time to operation. Incorrect diagnoses included myocardial infarction, renal calculi, pyelonephritis, and diverticulitis. Delays ranged from 13 hours to 9 days. Three of the six patients died.

The length of time from onset of symptoms to arrival in the emergency department ranged from 2 hours to 4 weeks. Time from hospital admission to operation ranged from 0 minutes to 9 days, with a mean of 5.2 hours. Fifty-four percent of patients were operated on within 2 hours of arrival. Length of time from onset of symptoms to admission or to

Table	II.	Preoperative
patient	cha	aracteristics

Characteristic	Total	Nonsurvivors	Survivors	
Arrival SBP	$101.2 \pm 46$	$98.5 \pm 51$	$105.7 \pm 38$	
Cardiac arrest	21	20	1	
Loss of con- sciousness	29	26	2	
Acidosis pH 7.3	49	37	10	
OR Hypotension SBP 80	34	30	4	
Transfusion PRBC/24 hours	$14.0~\pm~9$	$15.8\pm8.9$	$12.0~\pm~9.1$	

OR, Intraoperative; SBP, systolic blood pressure; PRBC, packed red blood cells.

operation could not be statistically correlated to mortality or morbidity rates.

Twenty-one patients sustained a preoperative cardiac arrest; 20 died. Twenty-nine patients were noted to have lost consciousness before operative intervention, 12 of those patients had a concomitant cardiac arrest. Twenty-six of these 29 patients died. Forty-nine patients were acidotic with a pH <7.3 on arrival at the emergency department; 37 of the 47 patients died. Thirty-four patients were severely hypotensive on arrival in the operating room, with systolic blood pressure <80 mm Hg; 30 of the 34 patients died. Thirty-six patients required transfusion of >18 units of blood including intraoperative and postoperative transfusions within 24 hours of operation (Table II).

Postoperative complications are listed in Table III. Twenty-nine patients required reoperation, undergoing a total of 34 additional operative procedures. Nineteen patients underwent laparotomy: nine for bleeding, four for ischemic bowel, four for wound dehiscence, two for emergent cholecystectomy, and one for abscess. In four the reason for reoperation was not clear. Some patients required intervention for several complications.

The overall mortality rate was 64% (72/113). Death occurred in an approximately bimodal distribution. Twenty-six deaths (36.1%) occurred during operation, and an additional 16 (22.2%) occurred within the first 48 hours after operation. Twelve patients (10% of total patients) died between 48 hours and 2 weeks, and 18 died more than 2 weeks after surgery. The mean survival for these patients who died more than 48 hours after surgery was 24.6  $\pm$  22.9 days.

By use of multivariate analysis, early death was found to be related to coma and preoperative cardiac arrest (p < 0.01) (Table IV). Presence of acidosis

 Table III. Postoperative characteristics

Characteristic	Total	Nonsurvivors	Survivors
Complications			
Renal	43	34	9
Respiratory	32	22	10
Cardiac	14	12	2
Neurological	9	7	2
Lower extremity	7	5	2
ischemia			
Intestinal	5	3	2
ischemia			
Reoperation	29		
Laparotomy	19	12	7
Vascular	7	4	3
Tracheostomy/O	7	1	6
tube			
Other	1	0	1
ICU Stay	12.1 days	15.5 days	9.5 days
	(0-112)		5
Days on ventilator	8.1	11.8	5.2
•	(0-72)		

and operative hypotension approached but did not achieve statistical significance (0.05 .Early death was not related to age, gender, previoussurgery, history of cardiac disease, hypertension,diabetes, or pulmonary or renal disease. Neither theduration of symptoms nor time to the operatingroom could be correlated with mortality rates in thisstudy. Although transfusion of more than 18 units ofred blood cells was a significant predictor of death byunivariate techniques, its effect was lost in multivariate analysis.

Late deaths, more than 48 hours after operation, occurred at a mean of  $24.6 \pm 22.9$  days. Late death was not influenced by preoperative cardiac arrest, loss of consciousness, acidosis, hypotension, excessive transfusion requirements, age, gender, preexisting medical conditions, length of stay in the ICU, or length of time from arrival in the emergency department to the operating room. Multivariate analysis showed that both renal and respiratory failure were statistically related to late death (p < 0.05). Reoperation was significantly associated with late death by univariate analysis but not by multivariate techniques (Table V).

By use of logistic regression analysis, overall hospital deaths were only related to postoperative renal failure (p < 0.01). Although postoperative renal failure, cardiac arrest, loss of consciousness, acidosis, severe intraoperative hypotension, respiratory failure, reoperation, and transfusion requirements of more than 18 units of red blood cells in 24 hours were significant contributors to death when considered by univariate analysis, this effect was lost

Risk factor	Present no.	% Mortality	Absent no.	% Mortality	p value
Cardiac arrest	21	81%	96	26.0%	< 0.01
Loss of consciousness	29	72%	88	23.8%	< 0.01
Acidosis ( $pH < 7.3$ )	49	53%	65	21.5%	< 0.1
OR Hypotension $(BP < 80)$	32	62.5%	83	24.4%	< 0.1

Table IV. Multivariate analysis of early death

Table V. Multivariate analysis of late deaths

Risk factor	Present no.	% Mortality	Absent no.	% Mortality	p value
Reoperation	23	65%	51	29.0%	NS*
Respiratory	32	69%	42	19.0%	< 0.01
Renal	34	75%	40	7.5%	< 0.01
Arrest	4	75%	71	38.0%	NS*
Loss of consciousness	7	71%	65	35.0%	NS*
Acidosis	22	55%	51	33.0%	NS*
OR hypotension	12	41.6%	63	39.7%	NS*
Transfusion >18 units/24 hr	18	50%	56	36.0%	NS*

OR, Intraoperative.

\*Significance < 0.05.

when multivariate techniques were applied (Table VI). Preoperative renal disease and elevated blood urea nitrogen or creatinine before operation were uncommon and could not be associated with post-operative failure. Hypotension on admission or in the operating room and acidosis could not be related to postoperative renal failure. No other patient characteristic could be correlated with postoperative renal failure (Table VI).

In evaluating data on patients with postoperative renal failure more closely, oliguric or anuric patients were found to have an 80% late mortality rate (30/37), whereas patients with high-output renal failure had a 66% mortality rate (4/6), and patients with normal postoperative renal function had a mortality rate of 10% (5/48). Anuric or oliguric patients undergoing dialysis had a slightly lower mortality rate than those not undergoing dialysis (73% vs 100%).

The need for prolonged ventilatory support (>7 days) was associated with a 69% late mortality rate (22/32), compared with 34% (18/35) for patients without postoperative respiratory compromise. Furthermore, 89% of patients who died after more than 2 weeks after operation had respiratory failure.

# DISCUSSION

The persistently high mortality rates for patients admitted with RAAA have been the subject of much discussion. With the exception of a few series,<sup>8-10</sup> mortality rates range from 40% to more than 70% in most reports. These poor results, in the face of low

mortality rates associated with elective surgery, have prompted many authors to suggest that the extreme preoperative derangements seen in some patients with RAAA makes further improvements in survival unlikely. Initial hopes that rapid transport might improve survival were not borne out in a recent study from Seattle.11 The poor results reported by this group led them to suggest that some patients should not be offered operative intervention because of the low likelihood of survival. Although these statements may have some appeal in a time of "cost containment," we believe that further data are needed before such a position can be supported. The ethical implications of the suggestion by Johansen et al.<sup>11</sup> are troubling to many surgeons and might have profound consequences for patient care, if implemented. Any acceptance of this thesis is dependent on two assumptions: (1) that preoperative factors are responsible for most postoperative deaths, and (2) that these particular deaths are responsible for a large portion of the resource consumption associated with postoperative deaths.

Undoubtedly, the cost of caring for patients with RAAA is high. Johansen et al.<sup>11</sup> found that the average cost of care for patients with RAAA was \$23,000. Patients dying within 24 hours of operation averaged \$5200; excluding these patients, average costs were \$38,000. Pasch et al.<sup>12</sup> found a mean cost of hospitalization of \$18,223 for RAAA in 1980 to 1981, with a mean of \$27,144 in patients surviving more than 24 hours after operation. It is clear that late deaths account for most of the resource expenditure

Risk factor	Present no.	Percent of deaths	Absent no.	Percent of deaths	p value
Cardiac arrest	21	95%	96	53%	NS*
Loss of consciousness	29	90%	88	51%	NS*
Acidosis pH $< 7.3$	49	76%	69	45%	NS*
OR Hypotension	32	78%	83	54%	NS*
Transfusion $18 + units$	36	75%	79	54%	NS*
Reoperation	23	65%	68	38%	NS*
Respiratory failure	32	69%	59	39%	NS*
Renal failure	43	79%	48	10%	< 0.01

Table VI. Multivariate analysis of overall deaths

\*Significance < 0.05.

for RAAA. The question then becomes whether one can differentiate causes of early and late death after RAAA, and more specifically, whether the preoperative status predicts late deaths. In reviewing the literature, we were not able to find data addressing these questions and, therefore, decided to review our own experience. We felt that it was imperative to distinguish between preoperative, intraoperative, and postoperative influences on early and late deaths. The correlation of a preoperative factor with late death would be the only finding that could possibly justify a policy of routine nonoperative care for a subgroup of patients. We also examined factors potentially related to early death in an attempt to pinpoint areas needing improvement to augment survival of patients with RAAA. A weakness of many prior studies of RAAA deaths is the lack of differentiation between early and late deaths and the absence of correlation of factors separately with regard to early and late deaths.

We found that 42% of deaths in patients reaching a hospital with RAAA occurred more than 48 hours after operation, at a mean of more than 3 weeks. This is not surprising given data from prior studies.<sup>11,12</sup> The implication of these findings is obvious; economic savings will be affected only if causes of late death can be decreased or eliminated. A corollary position suggests that the "economic cost" of those dying within 48 hours of operation may be low enough to justify intervention even in the face of high mortality rates for these subgroups.

Severe hemodynamic instability before operation was associated with death. Cardiac arrest, loss of consciousness, acidosis, and severe operative hypotension are all indicators of inadequate organ system perfusion. Several of these findings have been noted by other authors to correlate with deaths.<sup>8,13-26</sup> The significant observation in this study involves the timing of death. These preoperative factors were most important in affecting early deaths. Kouchoukous et al.<sup>27</sup> found that hypotensive patients died within 48 hours of operation. We found 58% of patients to be hypotensive (pressure <100 mm Hg) on admission, with 26% severely hypotensive (pressure < 80 mm Hg). Hypotension on arrival was not found to be related to either operative, early, or late deaths. However, severe hypotension on arrival in the operative suite was associated with an increased operative mortality rate, but not with any increase in postoperative deaths. Isolated blood pressure determinants may not be indicative of the actual degree of shock, as in a study by Morris et al.<sup>28</sup> who did not find any relationship between hypotension and death. Response to resuscitative efforts may be more important. Patients who were hypotensive on arrival in the operating room were less likely to survive, although this single factor was not significant when multivariate techniques were used. The effect of severe hemodynamic instability on late death was minimized by a relatively small number of patients with these risk factors surviving the immediate perioperative period. Although it appears that the prognosis for patients with arrest and coma is bleak, this is less true of patients with acidosis and hypotension, and in no case is death a certainty. The appropriate treatment of these patients will remain a cause for debate, but conclusions based on potential economic impact must take into consideration the differences between early and late deaths. Our own prejudice is that most of these individuals are entitled to emergency operation, since those that survive the early postoperative period have a chance of survival no different from the group as a whole.

We were not able to obtain complete data on clamp time or clamp location in these cases and thus were unable to determine the effect of these variables on mortality rates. We evaluated transfusion requirements within 24 hours rather than operative blood loss or intraoperative transfusion because we felt it would be a more inclusive indicator of perioperative blood loss. Excessive transfusions within 24 hours and reoperation for visceral or lower extremity ischemia were also associated with an increased mortality rate by univariate but not multivariate analysis. Other studies indicate that excessive transfusions or blood loss have a high correlation with death.<sup>10,17,20,22,25,27,29</sup> These complications may be associated with technical problems at the time of operation such as venous injuries, embolization, prolonged clamp time, and visceral devascularization. We would support the contention of Crawford<sup>8</sup> and others<sup>29-31</sup> that intraoperative technical problems are important factors influencing postoperative survival. We believe that these problems occur more frequently than is recognized. It is our belief that aortic control should be obtained by the most senior surgeon available, dissection should be minimized, and thromboembolectomy performed as needed. Proper placement of the aortic clamp (supraceliac when necessary), use of a straight graft when possible, and minimal periaortic dissection may decrease these complications. The debate over early operation versus rapid transport to a "vascular center" is not likely to be easily resolved. However, it seems clear that the operation should be performed by the most experienced surgeon available.8,32,33

Late deaths constituted 42% of our overall deaths and were related to organ system failure – specifically renal and respiratory failure. The overriding importance of these factors was made clear by multivariate analysis, in which renal failure was found to be the only predictor for overall deaths. We were unable to demonstrate any effect of preoperative status on specific postoperative complications. Late deaths are costly as far as human, technical, and economic resources are concerned. Reductions in deaths for these patients are likely to have the greatest economic impact. In contrast to some studies<sup>16,17,32</sup> we were unable to correlate preoperative renal insufficiency with postoperative failure. Several studies report increased mortality rates (70% to 100%) in patients with postoperative renal failure.<sup>19,20,23,34,35</sup> We found an 80% mortality rate in oliguric or anuric patients and a 66% mortality rate in patients with high-output renal failure. Patients with normal renal function sustained only a 10% mortality rate in comparison. Postoperative renal failure was not predicted by any preoperative characteristic examined. The trend toward improved mortality rates with dialysis suggests the need to consider a more aggressive approach in patients with renal dysfunction including earlier dialysis. The role of early dialysis or continuous arteriovenous hemofiltration to enable the maintenance of a more desirable hemodynamic situation

and early nutritional supplementation remains to be defined by further clinical studies.

Respiratory failure was present in a high percentage (89%) of our patients who died late in their hospital stay. Deaths further increased when both renal and respiratory failure coexisted. Tillney et al.<sup>36</sup> found respiratory failure to be the leading cause of death after operation in patients with RAAA. Respiratory failure was not predicted by preoperative or intraoperative characteristics examined, including a history of chronic obstructive pulmonary disease, smoking, or respiratory disease. Other studies have also found increased deaths with respiratory failure.<sup>15,20,22</sup>

Age has been suggested to be significant by some authors;<sup>10,14,16,19,27</sup> however, most studies find no statistical correlation between age and death or morbidity for ruptured aneurysms.<sup>9,13,17,29,30,32,37</sup> Age has been described as a significant risk factor for elective aneurysm repair in some series.<sup>21</sup> Our review found no correlation between age and death.

Review of the literature suggests that preoperative hypotension, preoperative cardiac arrest, free rupture, and preoperative anuria are the most predictive factors for death with rupture. However, the relationship of these factors to the timing of deaths is not well documented. Unlike elective repair, age, gender, cardiac history, chronic obstructive pulmonary disease, hypertension, and diabetes are not related to any increase in mortality rates. Time from admission to surgery was not a factor in mortality rates in this study although most of our patients were operated on within 2 hours. The large variation in duration of symptoms and time from hospital admission to the operating room suggests that prompt recognition of the symptomatic aneurysm remains a problem. For most of the patients in whom delay occurred, the diagnosis of RAAA was unsuspected by the admitting physician. This is discouraging and emphasizes the continued need to educate primary care and emergency physicians about the importance of this entity in the differential diagnosis of patients with abdominal or back pain.

Although the mortality rate after RAAA is high, multiple factors contribute to this. No doubt the patients' preoperative status has a strong effect on outcome, but it appears that intraoperative and postoperative events may be of equal or greater importance. Although we can do little to influence the condition of patients with RAAA before they reach the hospital, considerable potential exists to alter intraoperative and postoperative events. Furthermore late deaths that consume the most resources in patients with RAAA are not predicted by preoperative factors such as hypotension; nor do preexisting medical conditions significantly affect late deaths in a large series of patients. Late death is associated with organ system failure. Future efforts should be directed toward innovative strategies in this area. The timing of death after RAAA varies widely, and future reports on RAAA should consider this variable. Obviously, some patients will die despite maximal care. However, most of these patients, that is, patients with cardiac arrest, will die very early in their hospital course, consuming limited amounts of resources.

At this time, insufficient data exist to justify withholding operation from any group of patients based on economic resources. No factor was 100% predictive for death in our series.

Expeditious operative intervention with improved operative and postoperative care offers the best management for these patients. Efforts to decrease deaths and improve the cost/benefit ratio will involve the avoidance of technical problems, and include efforts to improve renal failure, respiratory failure, and multisystem organ failure.

#### REFERENCES

- Dubost D, Allary M, Oeconomos NA. Preoperos du Treatment des aneurysmes de l'aorta. Arch Surg 1952;64: 405-8.
- Gaylis H, Kessler E. Ruptured aortic aneurysms. Surgery 1980;87:300-4.
- Armour RH. Survivors of ruptured abdominal aortic aneurysm: the iceberg's tip. BM J 1977;2:1055-7.
- Ingoldby CJH, Wajanto R, Mitchell JE. Impact of vascular surgeon on community mortality from ruptured aortic aneurysms. Br J Surg 1986;78:551-3.
- Johansson G, Swedenborg J. Ruptured abdominal aortic aneurysms: a study of incidence and mortality. Br J Surg 1986;73:101-3.
- Hoffman M, Avellone JC, Plecha FR, et al. Operation for ruptured abdominal aortic aneurysms: a community-wide experience. Surgery 1987;91:597-602.
- Thomas PRS, Stewart RD. Abdominal aortic aneurysm. Br J Surg 1988;75:733-6.
- Crawford ES. Ruptured abdominal aortic aneurysm: an editorial. J VASC SURG 1991;13:348-50.
- Chiarello L, Reul GH, Wakasch DC, et al. Ruptured abdominal aortic aneurysm: treatment and review of eightyseven patients. Am J Surg 1974;128:735-8.
- Lawrie GM, Crawford ES, Morris GC, Howell JF. Progress in the treatment of ruptured abdominal aortic aneurysms. World J Surg 1980;4:653-60.
- Johansen K, Kohler TR, Nicholls SC, Zierler RE, Clowes AW, Kazmers A. Ruptured abdominal aortic aneurysm: the Harborview experience. J VASC SURG 1991;13:240-7.
- Pasch AR, Ricotta JJ, May AG, Green RM, DeWeese JE. Abdominal aortic aneurysm: the case for elective resection. Circulation 1984;70(suppl I):1-I4.
- Coles JC, Buttigliero J, Fisher G. The emergency abdominal aneurysm. Arch Surg 1966;93:6-9.

- Ottinger LW. Ruptured arteriosclerotic aneurysms of the abdominal aorta: reducing mortality. JAMA 1975;233:147-50.
- Levin PM, Shore EH, Treiman RL, Foran RF. Ruptured abdominal aortic aneurysms: surgical treatment. West J Med 1975;123:431-5.
- Graham AL, Najafi J, Dye WS, et al. Ruptured abdominal aortic aneurysm. Arch Surg 1968;97:1024-31.
- Wakefield TW, Whitehouse WM, Wu SC. Abdominal aortic aneurysm rupture: statistical analysis of factors affecting outcome of surgical treatment. Surgery 1982;91:586-96.
- Butler JJ, Chant ADB, Webster JHH. Ruptured abdominal aortic aneurysms. Br J Surg 1978;65:839-41.
- Hicks GL, Eastland MW, DeWeese JA, et al. Survival improvement following aortic aneurysm resection. Ann Surg 1975;181:863-9.
- Makin GS. Some factors influencing hospital mortality in ruptured abdominal aortic aneurysms. J Cardiovasc Surg 1983;24:646-8.
- McCabe CJ, Coleman WS, Brewster DC. The advantage of early operation for abdominal aortic aneurysm. Arch Surg 1981;116:1025-29.
- Sink JD, Myers RT, James PM JR. Ruptured abdominal aortic aneurysms: review of 33 cases treated surgically and discussion of prognostic indicators. Am Surg 1976;42:303-7.
- Couch NP, Lane RFC, Crance C. Management and mortality in resection of abdominal aortic aneurysms. Am J Surg 1970;119:408-16.
- Yashar JJ, Indeglic RA, Yashar J. Surgery for abdominal aortic aneurysms. Am J Surg 1972;123:398-405.
- 25. VanHeeckeren DW. Ruptured abdominal aortic aneurysms. Am J Surg 1970;119:402-7.
- Stoney RJ, Wylie EJ. Surgical treatment of ruptured abdominal aneurysms: factors influencing outcome. Calif Med 1969;111:1-4.
- Kouchoukous NT, Levy JF, Butcher HR. Mortality from ruptured abdominal aortic aneurysm. Am J Surg 1967;113: 232-5.
- Morris PJ, Buxton BF, Flanc C. Ruptured abdominal aortic aneurysms presenting to a general hospital. Med J Aust 1975;1:555-8.
- 29. Hildebrand HD, Fry PD. Ruptured abdominal aortic aneurysm. Surgery 1975;77:540-4.
- Pilcher DB, Dvais JH, Ashikaga T, et al. Treatment of abdominal aortic ancurysm in an entire state over 7½ years. Am J Surg 1980;139:487-94.
- Hiatt JCG, Barker WF, Machleder HI. Determinants of failure in the treatment of ruptured abdominal aortic aneurysm. Arch Surg 1984;119:1264-8.
- Ouriel K, Geary K, Green RM, et al. Factors determining survival after ruptured aortic aneurysms: the hospital, the surgeon, and the patient. J VASC SURG 1990;11:493-6.
- Shumacker HB, Barnes DL, King H. Ruptured abdominal aortic aneurysms. Ann Surg 1973;177:772-9.
- Lagaaij MB, Terpstra JL, Vink M. Ruptured aneurysms of the abdominal aorta. J Cardiovasc Surg 1970;33:440-6.
- Darling RC. Ruptured arteriosclerotic abdominal aortic aneurysms: a pathological and clinical study. Am J Surg 1970;119:397-401.
- 36. Tillney NL, Bailey GL, Morganh AP. Sequential system failure after rupture of abdominal aortic aneurysms: an unsolved problem in postoperative care. Ann Surg 1973;178: 117-22.

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### DISCUSSION

**Dr. Elizabeth Harrington** (New York, N.Y.). Dr. Harris and colleagues have reexamined a topic that has generated controversy at recent vascular society presentations. I congratulate her on her analysis of this extensive operative experience and on the idea that different factors may contribute to operative early and late deaths after RAAA.

The stated purpose of this paper is to analyze the causes of morbidity and death after surgery for ruptured aneurysm and to determine whether it is justified to withhold operation based on a patient's preoperative condition, as was suggested by Johansen's group at last year's ISCVS meeting. Dr. Harris attempts to demonstrate that most of the expense that society incurs with unsuccessful treatment of RAAA is due to late deaths that are not related to preoperative factors but to operative and postoperative complications. Since preoperative status cannot predict which patients will have expensive late deaths, Dr. Harris concludes that withholding operation for ruptured aneurysm is not ethically justified.

I agree with Dr. Harris that only rarely should treatment be withheld because of preoperative factors. Although Dr. Johansen suggested that no operation for RAAA be performed in a patient who had a preoperative cardiac arrest, making the decision to withhold treatment after this complication more difficult. Dr. Harris' group had a survivor after cardiac arrest.

Dr. Harris concludes that expeditious intraoperative intervention with improved operative and postoperative care offers the best management for these patients. In this regard I would like to ask her the following questions. First, your paper suggests that significant resuscitation was often given. What do you suggest regarding preoperative resuscitation? You suggest that the most senior surgeon available should obtain proximal control of the aorta. Who performed the operations in your series: surgical residents, vascular fellows, general surgery attendings, vascular attendings? How many different surgeons operated, and did surgical experience or training appear to influence the outcome? You state that your findings support the contention that intraoperative technical problems are important factors influencing postoperative survival. Can you state what technical problems occurred in the operating room? How do you recommend that control on the aorta be achieved? Finally, you suggest in your paper that early dialysis may help patients survive postoperative renal failure. Was there any evidence in your analysis that patients who had dialysis early did better than others? Do you have any other ideas on how to reduce deaths from postoperative renal failure and pulmonary failure?

Dr. Linda Harris. First of all, we do not intentionally maintain patients in a hypotensive state before operation; however, we try to get the patients to the operating room as expeditiously as possible. More than 50% of our patients were in the operating room within 2 hours of arrival. We have not specifically tried to examine the protocol of maintaining the patients' blood pressure at 50 to 70 mm Hg before operation, as suggested by Dr. Crawford. It is certainly possible that making the patient normotensive may loosen the clot and allow excessive bleeding as he has suggested.

In our particular series, the attending vascular surgeon is the person to obtain control of the aorta. We are a teaching institution, however, and the surgery itself is a combined effort of the senior surgical resident and the attending vascular surgeon. We did not look specifically at technical problems that occurred, but they would include venous injuries from dissection around the aorta, and delay to obtaining control of the aorta.

With regard to dialysis, we found that patients who were oliguric or anuric had only a 75% mortality rate when on dialysis, versus a 100% mortality rate for those not on dialysis. We did not examine early dialysis. Most patients in our series had dialysis beginning at 5 to 7 days after operation. Early recognition of renal failure might enable better survival for these patients. Most were maintained on routine dialysis and not continuous, which again, might provide a better hemodynamic environment and improve survival. These are all areas for future investigation.

Dr. Wiley Barker (Los Angeles, Calif.). Almost 30 years ago Jack Cannon, Joe Van DeWater, and I reported a series of 100 aneurysms, and our results were terrible. Sixty-five percent of our patients at that point were operated on electively, 35% came in ruptured, and I believe we had a 13% mortality rate in the patients with unruptured aneurysms, and approximately two thirds of the patients with ruptured aneurysms died. We thought we really were fighting a bad uphill battle operating on ruptured aneurysms, and we more or less adopted the policy that Dr. DeBakey has preached, in spite of some negative comments from Southern California from Dr. Berstein and his group, and chose to operate on almost every aneurysm we saw. The concept was that the aneurysm never gets smaller and is never less likely to rupture and the patient never gets younger.

And as we followed this in a couple of successive series of 100 patients-the last time I looked this up, we were operating on 91% of our patients in the overall spectrum of aneurysms as elective aneurysms-and by operating electively under the best circumstances possible, we were down to a mortality rate of between 2% and 3%. And the 9% ruptured aneurysms that we still dealt with were that group that you indicated who had been turned down-for instance, we have right now a very well-known senior surgeon of 99 years of age who has a 10 cm aneurysm and we are not about to operate on him. But that 9% constituted the patients who were very bad risks or the patients who really had not been identified before. And we now still lose approximately 55% or 60% of them, which boils down to the fact that now instead of losing what we had before, which was about 25 or 30 patients out of 100,

we are losing 6 or 7 out of the 100 out of the overall spectrum.

I think your points are very well made. It is nice to have an experienced hand around, but sometimes you cannot wait for that experienced hand.

**Dr. Harris.** Despite the fact that the absolute number of elective aneurysms repaired has increased, communitywide studies have shown that the actual number of ruptured aneurysms has not been decreased. This may be due, in part, to the increasing age of the population. Obviously, we still advocate elective repair whenever an aneurysm is recognized and the patient is a suitable candidate for surgery.

**Dr. Jesse Blumenthal** (New York, N.Y.). I have a comment and a couple of questions. First of all, many of us who review malpractice cases for our own company have always seen problems when there appears a 1-hour delay in treatment. These people are very litigious, and the \$5000 spent in trying to treat that patient is a lot less than the amount it takes to defend the surgeon under some of those circumstances, because under the current statutes, if a patient is denied any attempt at salvage, this is almost tantamount to a malpractice suit. So I think it is certainly the rare patient who should not be treated.

Now, in your figures you first said that there were 130 patients considered and 117 reviewed. Did any of those not undergo operation, or just why were those not included? Do you have any data on the delay in diagnosis in the emergency department?

Is there any effort at intraoperative autotransfusion in your series, and have you noticed any influence on that? Over the years of this study have you seen earlier dialysis having any effect? Certainly I think people who remember ruptured aneurysms 10 or 15 years ago, remember that the patients all died of renal failure. We have put patients on dialysis within 24 or 48 hours, and I think those are the ones that we have saved.

Dr. Harris. One hundred thirty patients did seek treatment, and 13 were not operated on because of patient or family refusal, and were, therefore, excluded from analysis. One of those patients had a carcinoma, and two were in their 90s.

Second, a delay in diagnosis occurred in six patients. These patients did not have a significantly different deaths than patients who were operated on promptly. This may be due to this subset of patients being more hemodynamically stable; this may also be a factor in the missed diagnosis. In other studies looking at delays and time to operation, what appears surprising to many is that patients operated on later do not have a higher mortality rate than those operated early. Usually misdiagnosis is attributed to cardiac or renal causes. We do use autotransfusion, and we have not examined its influence on deaths.

We are trying to initiate earlier dialysis. We did not particularly study how rapidly dialysis was performed over the time span of the study. We are beginning to use dialysis more liberally than we had in the past, but are somewhat limited by our colleagues in renal. What impact that will have on death remains to be seen.

**Dr. Ian Samson** (Lakewood, N.J.). I am a little troubled by your conclusion that preoperative hypotension is not responsible for late death. Certainly preoperative hypotension is responsible for renal failure, which obviously is a cause for late death. I wonder if you would comment on that, please.

**Dr. Harris.** In our study we were not able to correlate preoperative hypotension with renal failure. Intuitively, it seems to be related, but statistically, it could not be correlated. We were also unable to correlate other preoperative factors such as acidosis and loss of consciousness with postoperative complications.

Dr. John Ricotta. We were concerned about what Dr. Johansen implied, that is: if we can figure out the patients that are predestined to die, we will not operate on them and our mortality rate will go down. That is a little bit disturbing. I think that we have shown, as have others, that many of these people die late, and the ones that are predestined, if you will, all die early. The reason we could not find a correlation of hypotension with overall death is that people with hypotension died early and were not around to die late. We found that there were a lot of late deaths in people who had more than 18 units of blood transfused. Now, we are assuming that when you have more than 18 units of blood transfused there has been a technical problem. I think that generally is the case. Also when they are reoperated on for distal ischemia, vascular ischemia, or intestinal ischemia, it usually means that something has been thrown distally. So that is the assumption that we have made.

Because this is a retrospective study, we have not been able to change our activity to date, but I think we would agree that certainly we have to look at not only getting the patients to the operating room quickly, but also examine the way we take care of them in the operating room and the way we take care of them afterwards. We are trying to push our nephrologists to put them on dialysis early. It is very hard for us to get early dialysis from our nephrologists. It takes usually about 3 or 4 days of medical management before we can get the dialysis started.

I think the point of the paper was that there is still a lot we can do to reduce the number of deaths that we have control over, and we need to start to focus on that rather than trying to exclude patients from operation.